A COMPARATIVE STUDY OF CORONARY ANGIOGRAPHIC PROFILE WITH POSITIVE EXERCISE TREADMILL TEST IN PATIENTS WITH DIABETES AND NON-DIABETES

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CERTIFICATE

This is to certify that the dissertation entitled **"A COMPARATIVE STUDY OF CORONARY ANGIOGRAPHIC PROFILE WITH POSITIVE EXERCISE TREADMILL TEST IN PATIENTS WITH DIABETES AND NON-DIABETES"** is the bonafide original work of **DR.R.GUNASEKARAN** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2008.The period of post-graduate study and training was from August 2005 to July 2008.

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DECLARATION

I **Dr. R. GUNASEKARAN,** solemnly declare that this dissertation entitled,**"A COMPARATIVE STUDY OF CORONARY ANGIOGRAPHIC PROFILE WITH POSITIVE EXERCISE TREADMILL TEST IN PATIENTS WITH DIABETES AND NON-DIABETES"** is a bonafide work done by me at the department of Cardiology, Stanley Medical College and Government Stanley Hospital during the period 2005 – 2008 under the guidance and supervision of the Professor and Head of the department of Cardiology of Stanley Medical College and Government Stanley Hospital, **PROFESSOR DR. R. SUBRAMANIAN M.D., D.M.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place : Chennai

Date: 02.06.2008

Dr.R.GUNASEKARAN

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CONTENTS

INTRODUCTION

Cardiovascular diseases account for approximately 75% of the deaths that occur in patients with diabetes. People with diabetes have an increased prevalence of atherosclerosis and coronary heart disease and experience higher morbidity and mortality after acute coronary syndrome and myocardial infarction. Diabetes mellitus (DM) is a global health issue and burden. The population of DM patients is rapidly increasing in India, following the westernization of life the Indian style.

Diabetes Mellitus is an essential coronary risk factor and DM related coronary atherosclerosis is known for being more diffuse and progressive. As compared to Europeans diabetics from Asia have significant increase in the incidence of Coronary artery disease (CAD) as compared to non-diabetics. Because the clinical signs of coronary artery disease in diabetic patients are hard to detect and routine screening is costly, it would be of great benefit to try to either prevent CAD from occurring or to detect it early and provide optimal care by using a resting 12 Lead ECG, Exercise Treadmill Test and coronary angiography.

Exercise Treadmill Test is non-invasive relatively inexpensive and widely used in the clinical setting. Exercise tolerance testing is widely used as a diagnostic test in the initial evaluation of patients with symptoms suggestive of myocardial ischaemia and in persons with previously recognized coronary heart disease. Four major clinical uses of exercise testing relating to diagnosis, prognosis and functional assessment and therapeutic prescription.

Exercise stress testing is presently used to assess physical fitness determine functional capacity, diagnose cardiac disease, define the prognosis of known cardiac disease, prescribe an exercise plan and guide cardiac rehabilitation.

Coronary angiography (CAG) is presently the gold standard investigation in the evaluation of coronary artery disease. CAG, albeit a safe procedure, may cause serious complications especially, in patients with left main stenosis (LMS) .In our study its correlation with positive exercise treadmill test in patients with diabetes and without diabetes is assessed.

AIM OF THE STUDY

- To compare the diagnostic accuracy of exercise treadmill test in the detection of significant and severe Coronary artery disease as determined by coronary angiography between diabetics and non diabetics.

- To analyze and compare coronary anatomy and left ventricular function in patients with diabetes and without diabetes.

- To ascertain any difference in the clinical presentation and exercise treadmill test and angiographic profile of CAD between diabetic and non diabetic patients.

REVIEW OF LITERATURE

Coronary artery disease remains the leading cause of mortality and morbidity[1]. Earlier thought to be a disease of the modern world, it has been found to be equally or even more prevalent in underdeveloped and developing countries. This disease is unfortunately being witnessed in the younger population also.

DIABETES AND CORONARY ARTERY DISEASE

EPIDEMIOLOGY

Diabetes is the 7th leading cause of death in India, with much of that mortality a result of cardiovascular disease[2]. Ultimately atherosclerosis accounts for 65-80% of all deaths among South Indian patients with diabetes. A two to four fold excess in mortality due to CAD among the individual with diabetes has been noted in a number of prospective studies encompassing a variety of ethinic and racial groups[3].

Both type I and type II diabetes are therefore powerful and independent and risk factors for CAD. Although diabetes may be a problem of glucose metabolism, the American heart association has recently stated that diabetes is a cardio vascular disease.

PREVALENCE AND RISK FACTORS FOR CAD IN TYPE I DIABETES

Type I diabetes has seen that the first cases of clinically manifest CAD occur later in 3rd decade or in the 4th decade of life regardless of whether diabetes developed early in childhood or during late adolescence. CAD risk increases rapidly after the age of 40 and by the age of 55 years[4]. 35% of men

and women with Type I diabetes die of CAD. **KROLEWSKI ETAL** reported that angiographic studies have shown

that almost all patients with diabetic nephropathy older than age 45 have one or more clinically significant coronary stenosis[3]. Recent studies have shown that an association between angiotensin converting enzyme insertion / deletion (ACE I/D polymorphism) has been linked to MI in patient without diabetes as well as in patient with Type I and Type II diabetes.

PREVALENCE AND RISK FACTORS FOR CAD IN TYPE II DIABETES

Type II diabetes increases in relative risk of cardiovascular disease two to fourfold compared with risk in general population. The increase in cardio vascular risk is particularly high in women. The protection against atherosclerosis in pre-menopausal women is almost completely lost in women with diabetes. Many of these patients with Type II diabetes Mellitus have several risk factors for CAD. The term metabolic syndrome was first used by Gerald Reaven in 1988 to describe clustering of risk factors including hypertension, dyslipidemia, and hyperglycemia and insulin resistance.

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS[3].

In 1997, the American Diabetes Association proposed new criteria for the diagnosis of diabetes mellitus. These criteria use a Single blood glucose determination after 8 hour test (fasting plasma glucose, FPG), as the major diagnostic criteria.

Normal Value	Impaired fasting glucose level	Diabetes Mellitus
FPG < 110 mg/dl	FPG ≥ 110 mg/dl. And < 126mg/dl(IFG)	FPG ≥ 126mg/dl.
2 hour PG < 140 mg/dl.	2 hour PG more than 140 mg/dl. and < 200mg/dl (IGT)	2 hour PG ≥ 200 mg/dl.
2 hour PG 2 hour post load glucose		Symptoms of DM at random plasma glucose concentration ≥ 200 mg/dl.

DM = Diabetes Mellitus FPG = Fasting Plasma Glucose

IFG = Impaired fasting glucose IGT = Impaired glucose tolerance.

An FPG level less than 110/mg/dl. is considered normal. A new diagnostic category known as impaired fasting glucose (IFG) encompasses FPG greater than 110 but less than 126 mg/dl. An FPG higher than 126 mg/dl. establishes the diagnosis of diabetes mellitus.

Type II diabetes mellitus, previously known as non-insulin dependent or adult onset diabetes, represents 90% of the diabetic population Type I diabetes, known previously as insulin-dependent or juvenile onset diabetes, accounts for the remaining 10 percent.

A Finnish epidemiological survey compared the rates of myocardial infarction (MI) in non diabetic and diabetic population[1]. In this study, patient with diabetes but without prior myocardial infarction had the same level of risk for subsequent acute coronary event as non diabetics with a history of previous myocardial infarction.

PATHOPHYSIOLOGY OF DIABETIC CARDIOVASCULAR COMPLICATION

The increased risk in cardiovascular disease in individuals with diabetes is explained in part by the clustering of risk factors including dyslipidemia, hypertension, hyperglycaemia, hyperinsulinemia and prothrombotic factors.

INSULIN RESISTANCE & HYPERGLYCAEMIA

Insulin resistance that is present many years or more before clinical onset of overt diabetes resistance is associated with other atherogenic risk factors such as hypertension and a pro-coagulant state that promotes atherosclerosis many years before overt hyperglycemia ensues. Several studies have shown an inverse correlation between insulin sensitivity and atherosclerosis.

THE BRUNECK STUDY DATA BASE suggests these risk factors are present in 84% of patients with type II diabetes[2]. `Thus an increased prevalence of CAD is apparent in patients with impaired glucose tolerance and in those with newly diagnosed Type II diabetes. The duration of insulin resistance among hyperglycaemic and diabetic individuals probably contributes to the development of atherosclerosis.

However, no obvious association between the extent or severity of macro vascular complication and duration are severity of Type II diabetes has been found most likely because the duration of insulin resistance is often unknown. Serum glucose level may be an important risk factor for CVD. The direct relationship between glucose levels and cardiovascular disease also is seen in patients with Type I diabetes. A 1% increase in glycosylated hemoglobin doubled the increase in cardiovascular disease[3].

DYSLIPIDEMIA

An important mechanism for the development of diabetic atherosclerosis is dyslipidemia. The central feature of diabetic dyslipidemia is increased levels of VLDL due both to increased

production of VLDL and to decreased catabolism of triglyceride - rich Lipoproteins including chylomicron. Although the dyslipidemia of diabetes is not characterized by marked elevation of LDL. There are differences & LDL type found in patients with Type II diabetes. Specifically LDL is smaller and denser than typical LDL particles. These smaller, denser LDL particles have a greater tendency to undergo Oxidation which accelerate the atherosclerotic process.

ADVANCED GLYCATION END PRODUCTS IN DIABETES

AGEs occur as a result of the nonenzymatic glycation of both lipids and proteins[2]. Initially, labile covalent bond develops between the aldehyde of the glucose molecule and the amino acid side chain on both sugars and lipids. Specifically, glucose is covalently bound mainly to lysine residues in proteins, forming fructose-lysine residues. This reaction results in the development of Schiff base, which, in turn, undergoes another chemical reaction to form a ketomine, termed an Amadori product. These products result in cumulative oxidative damage to proteins. These products include CML and pentosidine.

The increased levels of pentosidine and CML correlate with the severity of diabetic complications, including nephropathy, retinopathy, and vascular disease. One such Amadori product is glycated (or glycosylated) hemoglobin A1c (HbA1c), which is commonly used to monitor glycemic control in diabetic patients. Since both free-radical oxidation and glycation are involved, these substances are also called glyoxidation products.

AGEs cross-link to the proteins composing the extracellular matrix and vascular basement membrane, which results in reduced solubility and decreased enzymatic digestion AGE formation also prevents proper assembly of basement proteins, thereby altering their function. This in turn may alter the ability of cells to bind to their substrates.

AGEs are derived from oxidation of lipids. Enhanced glycation, oxidation, and glyoxidation of lipoproteins have been postulated as a possible cause for the development of diabetic macrovascular disease. Certainly there are increased levels of AGE-modified LDL-apoprotein and LDL-lipid in persons with diabetes relative to levels in persons without diabetes.

Vlassara and colleagues identified a specific receptor for AGEs on monocyte/macrophages, termed RAGE (receptor for AGEs)[3]. The subsequent interaction with the AGE and its receptor may induce the release of the cytokines tumor necrosis factor (TNF) and interleukin-1. Other cytokines that have been demonstrated include the synthesis and release of procoagulant activity and platelet-activating factor (PAF) by endothelial cells, as well as the induction of platelet-derived growth factor (PDGF-AA), which can be indirectly responsible for fibroblast and smooth muscle proliferation. Further-more, increased AGE-receptor interaction has been shown to result in the enhanced expression of vascular cell adhesion molecule (VCAM), which in turn results in increased atherogenesis.

INCREASED OXIDATIVE STRESS IN DIABETES

This increased stress may be due in part to the decreased availability of antioxidants such as ascorbic acid, Vit-E, Uric acid and glutathiamine. In addition there may be increased lipid peroxidation products and superoxide anion products which may lead to altered vascular function.

THROMBOSIS AND FIBRINOLYSIS IN DIABETES

Plaque disruption with Overlying thrombosis is a major cause of acute coronary syndrome including MI, sudden death, stroke. Because patient with Type I & Type II diabetes particularly to those with Type II Diabetes have higher rates of acute coronary syndromes than the population without diabetes, heightened increased procoagulant activity or arterial prothrombotic reactivity may play a pivotal role in development of this macro vascular complication. Fibrinolysis also impaired in individuals with diabetes particularly, those with Type II diabetes.

There are three underlying mechanisms for this prothrombosis: heightened platelet reactivity, increased procoagulant activity, and decreased antithrombotic and fibrinolytic activity. The principal components of a thrombus are platelets and fibrin. The coagulation is initiated by the exposure of tissue factor within the arterial plaque at the time of plaque disruption. This results in the activation of factor VII / VIIa, which forms the "tenase complex" with factors X and V, resulting in the activation of thrombin. Thrombin stimulates platelet reactivity and the conversion of fibrinogen to fibrin, producing a thrombus.

Patients with diabetes have increased concentrations of fibrinogen, von Willebrand factor, and factor VII. Although the mechanisms of the increased concentrations of these factors have yet to be elucidated, the level of serum fibrinogen correlates with the levels of proinsulin and insulin in the blood.

Several reports indicate that the activity of antithrombotic factors, including protein C and antithrombins, are decreased in subjects with diabetes, which further potentiates the hypercoagulable state. Fibrinolysis is also impaired in individuals with diabetes, particularly those with type 2 diabetes. This impairment may be due to the increased activity of PAI-1 in the blood, which counteracts the

action of native tissue plasminogen activator (t-PA) to induce fibrinolysis.

PAI-1 is elevated not only in resulting states but also in response to physiologic stimuli. The serum level of PAI-1 may be elevated as a result of several factors, including elevated serum levels of insulin, lipids, and glucose. The impairment of the fibrinolytic system can potentially exacerbate the development and persistence of thrombi, resulting in an increased risk of vascular occlusion.

EXERCISE TREADMILL TEST IN DIABETES[2]

The American Diabetes Association / American College of Cardiology (ADA / ACC) have developed a consensus as to which patients are at increased risk for cardiac events, which patients should be screened. Exercise testing in patients with diabetes is given a Type II classification by evidence or opinion. Although stress testing is clearly is indicated for those diabetic individuals who have established CAD, it is not clear what test would be the appropriate in patients who do not have CAD.

Following are indication get by ADA / ACC consensus panel.

1) Typical or atypical cardiac symptoms
2) Resting electrocardiography findings suggestive of ischaemia or infarction
3) Peripheral or carotid occlusive arterial disease.
4) Sedentary life style, age 35 years and plan to begin a vigorous exercise programme.
5) 2 or more of the risk factors listed below in addition to diabetes
    a) Total cholesterol more or equal to 240 mg/dl., LDL cholesterol more or equal 160 mg/dl., and or HDL cholesterol less than 35 mg/dl.
    b) Blood pressure more than 140/90 mm of hg

c) Smoking

d) Family history of premature coronary artery disease

Stress testing is warranted if the patient has typical or atypical cardiac symptoms. Silent ischemia is a frequent occurrence in diabetic individuals. A resting ECG showing evidence of an infarction warrants stress testing. The presence of peripheral or carotid occlusive arterial disease is strongly associated with CAD. Therefore any diabetic individual with evidence of peripheral vascular disease should undergo stress testing. The addition of two other risk factors to diabetes increases the cardiovascular death by three fold.

Asymptomatic patients with diabetes and one or no risk factors along with a normal ECG do not require cardiac testing. These patients with carotid or peripheral vascular disease, beginning a vigorous exercise programme, with minor ST-T wave changes on ECG or with two or more risk factors should undergo an exercise stress test.

CONTRAINDICATIONS TO EXERCISE TESTING[1]

ABSOLUTE

Acute myocardial infarction (within 2 days)

High risk unstable angina

Uncontrolled cardiac arrhythmias

Symptomatic severe aortic stenosis

Uncontrolled symptomatic heart failure

Acute pulmonary embolus or pulmonary infarction

Acute myocarditis or pericarditis

Acute aortic dissection

RELATIVE

Left main coronary stenosis

Moderate stenotic valvular heart disease

Electrolyte abnormalities

Severe arterial hypertension

Tachyarrhythmias or bradyarrhythmias

Hypertrophic cardiomyopathy and other forms of outflow tract obstruction

Mental or physical impairment leading to inability to exercise adequately

High-degree atrioventricular block.

INDICATIONS FOR TERMINATING EXERCISE TESTING[1]

ABSOLUTE INDICATIONS

Drops in systolic blood pressure of > 10 mm Hg from baseline blood pressure despite an increase in workload, when accompanied by other evidence of ischemia.

Moderate to severe angina

Increasing nervous system symptoms (eg. ataxia, dizziness, or near-syncope)

Signs of poor perfusion (cyanosis or pallor)

Technical difficulties in monitoring ECG or systolic blood pressure

Subject's desire to stop

Sustained ventricular tachycardia

ST elevation (> 1.0 mm) in leads without diagnostic Q-waves

(other than V1 or aVR).

RELATIVE INDICATIONS

Drop in SBP of (>10mm Hg from baseline BP despite an increased in work load, in the absence of other evidence of ischemia

ST or QRS changes such as excessive ST depression (> 2 mm of horizontal or downsloping ST-segment depression) or marked axis shift.

Arrythmias other than sustained VT, including multifocal PVCs, triplets of PVCs, SVT , heart block or bradyarrhythmias.

Development of BBB or IVCD that cannot be distinguished from VT.

Increasing chest pain. Fatigue, shortness of breath, wheezing, leg cramps,

Hypertensive response.

A negative test at a high work load defined as completing 9 minutes or stage 3 of a Bruce treadmill protocol, should provide some reassurancc of a favourable prognosis with regard to cardiovascular disease[3]. However, these patients must be routinely followed and should their symptoms change undergo a repeat stress test. Those individuals who have mildly positive test (defined as 1 to 1.5 mm ST Depression at a moderate to high exercise level (Bruce stage 3) are also in a low risk group[38].

A moderately positive test in an asymptomatic person with diabetes should warrant a stress echo or cardiac nuclear test, a large persistent defect indicates a significant risk for cardiac events in the next one to two years. If the defect is moderate or large as determined by echo or perfusion testing, the thresold for having the patients undergo cardiac catheterization should be low. A markedly positive stress test is defined as hypotension with exercise a positive test at a heart rate of less than 120 beats per min., an exercise capacity of less than 6 minutes. and greater than 2 mm ST depression[1]. The asymptomatic patients with diabetes who has such a test should undergo cardiac catheterization.

The exercise ECG result is more likely to be abnormal in patients with more severe coronary arterial obstruction, with more extensive CAD, and other more strenuous levels of exercise. Early onset of angina, ischemia ST segment depression, and fall in blood pressure at low exercise workloads are the most important exercise parameters associated with an advance prognosis and multi vessel CAD.

EXERCISE PARAMETERS ASSOCIATED WITH AN ADVERSE PROGNOSIS AND MULTI VESSEL CORONARY ARTERY DISEASE.

1. Duration of symptom limiting exercise less than 5 minutes.
2. Failure to increase systolic blood pressure more or equal to 120 mm are sustained decrease more or equal to 110 mm Hg. or below rest levels during progressive exercise.
3. ST segment depression more or equal to 2 mm down sloping ST segment, starting at < 5 METS, involving more or equal to 5 leads, persisting more or equal to 5

minutes into recovery.

4. Exercise induced ST Segment elevation (avR excluded)

5. Angina pectoris at low exercise workload

6. Reproducible sustained > 30 sec or symptomatic Ventricular tachycardia.

ECHOCARDIOGRAPHY - LV FUNCTION ASSESSMENT

Echocardiography can measure several parameters as an extension of systolic function of the heart. These parameters are LVEF Fractional shortening stroke volume and cardiac index, systolic tissue velocity of the mitral annulus and myocardial strain and regional wall motion analysis[1].

LEFT VENTRICULAR EJECTION FRACTION

The most well accepted expression of global LV Function is LVEF. As the term indicates, it is simple measure of how much end-diastolic volume is ejected from the LV with each contraction. Although it has many limitations, including load dependency, LVEF has been found to be a strong predictor of clinical outcome in almost all major cardiac condition, and it is served to relative optimal management strategies. In clinical practice, LVEF is frequently determined by "eye balling" 2D Echocardiographic images of the LV. This visual assessment is reasonably reliable when performed by an experienced Echocardiographer but varies widely among readers. Therefore LVEF should be measured more objectively whenever possible, using volumetric measurements as described by the following equations.

LVEF = (LVEDV - LVESV) / LVEDV.

Where LVEDV and LVESV are LV end-diastolic volume and end-systolic volume respectively.

LVEF can also be calculated from LV dimension measured with m-mode or 2 D Echocardiography, M mode or 2D Echocardiographic measurement of LV dimension from the mid ventricular level is used to calculate LVEF as follows[1] :

$$LVEF = (LVEDD^2 - LVESD^2) / LVEDD^2$$

Where LVEDD & LVESD are end diastolic dimension and end systolic dimension respectively. This equation is actually percentage change in LV area, or fractional shortening of the LV short axis, which equals LVEF if the apical long axis dimension remains the same from the diastolic phase to systolic contraction. Because the apical long axis normally shorten 10 to 15 percent with systolic an apical correction factor is added on the basis of contraction of the apex. 5 to 7 percent for normal to hyperdynamic apical contraction, 3 percent for hypokinetic contraction, and 0 percent for akinetic apex.

FRACTIONAL SHORTENING

Fractional Shortening (FS) is a percentage in LV dimensions with each LV contraction.

FS = (LVEDD - LVESD / LVEDD.

This systolic function parameter is now rarely used for diagnosis or clinical decision making.

ANGIOGRAPHIC FEATURES OF CORONARY ARTERY DISEASE IN PATIENTS WITH DIABETES

Angiographic studies have documented the severe and diffused nature of atherosclerotic coronary artery involvement in patients with diabetes[3]. The patients with diabetes have a greater number of coronary vessels involved with more diffused distribution of atherosclerotic lesion. Large angiographic studies comparing patients with diabetes to matched controls in the setting of acute MI or elective angioplasty or prior to coronary bypass surgery have all shown that diabetes is associated with significantly more severe proximal and distal CAD[51].

An important finding with regard to pathogenesis of acute coronary syndromes angiographic evidence suggesting a significant increase in plaque ulceration and thrombosis in diabetic compared with non diabetic patients.

The diffuse nature of coronary atherosclerosis in diabetes may contribute to systolic dysfunction of the non infracted myocardium. Moreover a recent study has shown that patients with diabetes have reduced ability to develop collateral blood vessels in the presence of CAD, a finding that also may explain in the more frequent occurrence of post infarction angina and infarct expansion[3].

Patient with diabetes surviving MI also suffer higher case mortality rates than patients without diabetes. Case mortality is related primarily to both recurrent MI and the development of new congestive heart failure.

CORONARY ARTERY STENOSIS - ANGIOGRAPHIC ASSESSMENT

An angiographic lumen narrowing is commonly referred to as a stenosis[53]. It may be due to atherosclerosis vasospasm or angiographic artifact. It is necessary to quantify the coronary stenosis accurately it must be seen in profile, free from artifact related to foreshortening or obstruction by a crossing vessels. Therefore multiple views are important. When seen across its major axis, the width of the lumen may appear to be normal, but a clue to the presence of a severe degree of narrowing in the other axis, may be marked lucency caused by thinning of contrast lumen.

The normal caliber of major coronary arteries left main 4.5 plus or minus 0.5 mm, LAD 3.7 plus or minus 0.4 mm, LCX 3.4 plus or minus 0.5 mm for non-dominant and dominant LCX 4.2 plus or minus 0.6 mm, RCA 3.9 plus or minus 0.6for dominant and 2.8 plus or minus 0.5 mm for non-dominant.

The evaluation stenosis relates to the percentage of reduction in diameter of the vessel size. The diameter stenosis is calculated in the projection where the greatest narrowing is seen. By comparing the diameter of a presumably disease free segment of coronary artery to the size of the diagnostic catheter (6 F equals to 2 mm). The operator can identify vessels that fall below this normal size ranges and may thus be diffusely diseased.

It should be noted that the stenotic lumen is compared to nearby unobstructed lesion which

indeed may have diffuse atherosclerotic disease and thus is angiographic normal, but still may be diseased. The area of stenosis is always greater than diameter stenosis and assumes the lumen is circular. Whereas, most of the time the lumen is eccentric. A 50% reduction in diameter is equivalent to a 75 reduction in cross sectional area, and a 75% reduction diameter is equal to a 90% reduction in cross sectional area. Stenosis that reduce the lumen diameter by 50% (and hence cross sectional area by 75%) is haemodynamically significant.

Because of the subjective nature of a visual lesion assessment, there is a plus or minus 20% variation between readings of two or more experienced angiographers especially for lesions 40 to 70% narrowed. The simplest way to resolve this problem is to project the coronary image on a wall mounted viewing screen and use inexpensive digital calipers to measure the relative diameters of stenotic and reference segment. Percent stenosis can be calculated as 100 x (1 - stenosis diameter / reference diameter) to provide a more accurate estimate of stenosis.

TIMI FRAME COUNT

Myocardial blood flow has been assessed angiographically using thrombolysis in myocardial infarction (TIMI) score for qualitative grading of coronary flow[53]. TIMI flow grade 0 - 3 have become a standard definition of angiographic coronary blood flow in clinical trials. Quantitative method of TIMI uses cine angiography with 6 F catheter and filming at 30 frames per second. The number of cine frames form the introduction of the dye in the coronary artery to predetermined distal landmark is counted.

The first frame used for TIMI frame counting is that in which the dye fully opacifies the artery

origin and in which the dye extends across the width of the artery touching both borders with anti grade motions of the dye. The last frame counted is when the dye enters the first distal landmark branch. Full opacification of distal branch segment is not required..

The TIMI frame count has can be corrected further the length of the LAD. The TIMI frame count in the LAD artery requires normalisation or correction for comparison with the two other major arteries. This is called corrected TIMI frame count. (CTFC). CTFC accounts for the difference the dye as to travel in the LAD relative to the other arteries. CTFC divides the absolute frame count in LAD artery by 1.7 to standardize the distance of dye travel in all the three arteries. The average LAD coronary artery is 14.7 cm long, RCA 9.8 cm. LCX 9.3 cm.. Normal TFC for LAD is 36 plus or minus and CTFC 21 plus or minus 2, for LCX TFC is 22 plus or minus 4 for RCA , TFC is 20 plus or minus 3. TIMI flow grades do not correspond to measure doppler flow velocity or CTFC. High TFC were associated with micro vascular dysfunction despite an open artery. CTFC of less than 20 frames were associated with low risk for adverse events in patients after myocardial infarction. A contrast injection rate of more than 1 ml/sec by hand injection can decrease the TIMI frame count by 2 frames. The TIMI frame count method provides valuable information relative to clinical responses after coronary interventions.

CORONARY COLLATERAL CIRCULATION

The reopacification of a totally (or) sub totally (99%) occluded vessel from antigrade (or) retrograde filling is defined as collateral filling[53] Angiographically visible colleteral channels are not usually seen until the coronary obstruction is greater than 90% at which point coronary perfusion pressure falls substantially and the blood flow through the collaterals increases. The collateral circulation may provide upto 50% of antegrade coronary flow into chronic total occlusion.

Networks of small anastamotic branches interconnect the major coronary arteries and serve as precursors for the collateral circulation that maintains myocardial perfusion despite the development of severe proximal atherosclerotic narrowing. Collateral channels may not been seen in patients with normally or mildly diseased coronary arteries because of their small (<200/mic.mtr.) caliber, but as CAD progresses and becomes more severe (> 90% stenosis), a pressure gradient is generated between the anaestemotic channels and the distal vessel that is hypoperfused. The transstenosis pressure gradient facilities blood flow through the anaestemotic channels, which progressively dilate and eventually become visible as collateral vessels.

PIONEER WORKS OF CORONARY ANGIOGRAPHY CORRELATION WITH POSITIVE EXERCISE TREADMILL TEST IN DIABETICS AND NON-DIABETICS.

In chronological order **Alen G.Bartel** established the angiographic severity of coronary artery disease correlates strongly with the frequency of positive exercise stress test[13]. Left main coronary stenosis of 70% or greater was associated with more severe ST segment changes, inability to achieve target heart rate during stress, and lower maximum heart rate during stress. He demonstrated the angiographic occurrence of collateral vessel was related to the extent of coronary artery disease and was associated with high percentage of positive exercise tests.

Later, **Jaishankar et al** reported that incidence of extensive Tripel vessel disease with probable diffuse disease is more in patients with diabetes[5]. They also suggested the extent of left ventricular

dysfunction associated with single vessel disease is more in diabetics and coronary artery disease with multiple vessels involvement is more common in patients with diabetes and they also have a greater incidence of diffuse disease.

Subsequent works by **Bagchi Sokmita** established that Atherosclerotic CAD is more severe in diabetics making diabetes an independent risk factor for macro vascular disease[9].

Girish K. Sonwalker revealed that Triple Vessel Disease was significantly common in diabetics and LAD was the commonest vessel involved. He also suggested that LMCA disease involvement in diabetics was common when compared with non diabetics i.e. 59% in diabetics[28].

Several studies have reported the diabetics patient express a pattern of multi focal significant atherosclerotic lesion[18]. Diabetes Mellitus results in a higher incidence of chronic total occlusion and more diffuse coronary lesion. It has also been shown that LMCA involvement was also higher in diabetics and majority of diabetics had Triple Vessel Disease and Complex lesion (Bifurcation) and calcification were significantly higher in diabetics[51].

MATERIALS AND METHODS

PATIENT SELECTION CRITERIA:

We have selected 40 number of CAD patients with diabetes were randomly selected for study (Group A) and results were compared with control of 40 CAD patients without diabetes (Group B)

They were men and women ranging in the age from 30 to 65 years with a mean of 49.10 years. All patients were evaluated with detailed history, examination and laboratory investigations including fasting blood sugar, postprandial blood sugar, lipid profile 12 lead ECG exercise treadmill test with Bruce Protocol and coronary angiography (CAG). They were included in the study after strictly adhering to study protocol.

EXCLUSION CRITERIA :

Recent myocardial infarction

Uncontrolled hypertension

Unstable angina

Congestive heart failure

Atrial fibrillation

Severe chronic obstructive pulmonary disease

Second and third degree AV Block.

Evidence of primary myocardial or valvular heart disease

CLINICAL DATA :

We have taken the blood pressure with a mercury device, height and weight of the each patient. All the blood tests were done after 12 hours of over night fasting by calorimetry. Patients already taking hypertensive medication or those whose average of two blood pressure readings atleast 5 minutes. apart in the sitting posture was greater or equal 140/90 mm of Hg. were labeled as hypertension.

Also patients with a history of taking anti hyperlipidemic drugs, total cholesterol more or equal to 200 mg/dl. or low density lipoprotein more or equal to 130 mg/dl. were defined as hypercholesterolemic. Diabetes Mellitus was diagnosed to be present if a patient had a definite history of diabetes with records of treatment or fasting plasma glucose more or equal to 126 mg/dl. or also two hour post load glucose more or equal to 200 mg/dl. based on the guidelines of the American Diabetes Association 2003. Angina Class of each patient was defined according to CCS Classification.

Myocardial infarction or sudden death before the age of 55 years in the father or any other male first degree relative or before the age of 65 years in the mother or any other female first degree relative was considered a positive family history of premature coronary artery disease (CAD). A smoker was defined as a person who regularly smoked cigarettes or who had stopped smoking within past one month.

EXERCISE TEST :

All the patients were exercised in the Treadmill according to the Bruce and modified Bruce Protocol under cardiologist's supervision. Patients were brought into the exercise laboratory two hours or more after its last meal. The exercise test procedure was explained to them and each patient's consent was obtained. A conventional 12 Lead resting ECG was performed prior to exercise with electrodes for the standard limb leads placed on the upper left chest, upper right chest and lower left and right abdominal wall in order to obtain a stable base line during exercise.

The patients were exercised according to the Bruce protocol[55], using the graded multistage treadmill test. Exercise was continued for three minutes. at each treadmill stage. (Stage I - 1.7 mph, 10% grade ; Stage II - 2.5 mph, 12% grade ; Stage III - 3.4 mph, 14% grade ; Stage IV - 4.2 mph, 16% grade ; Stage V - 5.0 mph, 18% grade) . Patients were encouraged to exercise to their maximum.

Exclusion Criteria for the exercise in this database were

1) Those who could not exercise for any reason
2) Those with significant valvular or congenital heart disease
3) Acute Myocardial Infarction - Recent onset (7 days)
4) Percutaneous or coronary surgery intervention
5) Decompensated heart failure
6) Advanced AV Block
7) High risk unstable angina

Metabolic equivalent (METS) was used to express the estimated work load[1]. The term METS

refers to unit of oxygen uptake in a sitting, resting person and one METS is equivalent to 3.5ml O2 / Kg./min. of body weight. Measured volume of oxygen in ml. of O2 /min/kg divided by 3.5 ml O2/kg/ min. determines the number of METS associated with activity. Exercise testing was stopped if exertional hypotension, malignant ventricular arrhythmias, marked ST segment depression more than 3 mm., ST elevation more or equal to 1 mm. in non-infarct leads without diagnostic Q waves (other than V1 or avR) and limiting chest pain were reported.

The positive exercise ECG was determined by conventional criteria more or equal to 1 mm of horizontal or down sloping ST segment depression at 80 m. sec. after the end of the QRS complex (from J point) in three consecutive beats. Exercise tests were interpreted as negative only if the patient achieved 85% of the predicted maximal heart rate at peak exercise (target heart rate) in the absence of significant ST changes. The tests was placed in the undetermined category if any of the following occurred.

1) The patient failed to achieve target rate in the absence of significant ST segment change
2) The patient had resting electrocardiographic abnormalities such as bundle branch block, precluding evaluation of ST segment at maximal stress.
3) Multiple premature beats or unstable base line activity at maximal heart rate obscured possible ST segment changes.

ECHOCARDIOGRAPHIC DATA :

Two dimensional and M-mode measurements were obtained with patients in left lateral position using an Aloka SSD 4000 phased array system equipped with Tissue doppler and Harmonic imaging technology with Doppler frequency of 2.5 to 3.8 MHz. With measurement of LV end diastolic

dimension in diastole and LV end systolic dimension in systole, LV Ejection fraction was measured.

CORONARY ANGIOGRAPHIC DATA :

Cardiac catheterization was performed less than or equal 90 days after the exercise test using standard technique and was recorded in multiple projections for the left and right coronary arteries. Coronary angiogram done in the Siemens mobile unit cath lab in our hospital. Coronary angiograms were done through right femoral approach using modified seldinger technique after getting patient's and patient's relative consent. Low osmolar non ionic contrast agent (Omnipaque) was used. Coronary angiography was performed with the judkins catheter after Injection of 2500 IU IV Heparin. Coronary artery stenosis was evaluated by use of multiple projection quantitative analysis was done with a medical imaging system CMS analysis software.

CAD was defined as the presence of more or equal to 50% stenosis in the coronary arteries. Single Vessel Disease (SVD) ; Stenosis of one of the left anterior descending artery or left circumflex artery or right coronary artery or main branches of each. Two Vessel Disease (DVD): Stenosis of two coronary arteries other than left main artery. Three Vessel Disease (TVD) - Stenosis in three coronary arteries other than left main artery and Left Main stenosis (LMS) ; Stenosis in Left main artery regardless of existence of stenosis in other arteries. Those patients with significant lesions in CAD with diabetes were Grouped as Group A. Those with significant lesions in CAD without diabetes were Grouped as Group B.

RESULTS AND DATA ANALYSIS

Datas are expressed as Mean Value plus or minus standard deviation (SD). Statistical significant analysis defined as $P < 0.05$ Statistical analysis was done using SPSS Software system. Based on the study protocol, 80 study patients were divided as those significant coronary artery lesion and positive exercise treadmill test in Diabetic patients were grouped as Group A (N = 40) and those without diabetes were grouped as Group B (N = 40) for statistical analysis. History obtained and physical findings in both groups did not show much difference. There was no difference in the distribution of the some of the variables like systolic blood pressure, diastolic blood pressure, pulse rate, history of drug intake like Isosorbide dinitrate, Aspirin, Enalapril Atenolol and dyslipedemia in both groups.

They were thoroughly evaluated by 12 Lead ECG, Exercise Treadmill Test, 2 D M-Mode Doppler and Tissue Doppler Echocardiography and Coronary Angiography. The results of the study were as follows :

Independent-Samples T Test

The Independent-Samples t test procedure is used to compares mean scores of two types of diabetic mellitus. The procedure assumes that the variances of the two groups are equal and it was tested with Levene's test statistics. Mean scores was tested with t test procedures and the results of the analysis are given in Table ---.

Null Hypothesis: H_0 : There is no significant difference between the mean scores of diabetic mellitus regarding with certain factors.

Table -----ANOVA – Diabetic mellitus * Variables

	DM Type	N	Mean	Std. Deviation	t	df	p value	Remark
SBP	Non Diabetic	40	129.25	11.694	1.072	78	.287	Not Significant
	Diabetic	40	132.25	13.276				
DBP	Non Diabetic	40	85.48	6.441	1.377	78	.173	Not Significant
	Diabetic	40	87.75	8.230				
PR	Non Diabetic	40	74.05	5.556	1.241	78	.218	Not Significant
	Diabetic	40	72.50	5.616				
FBS	Non Diabetic	40	88.80	6.806	17.056	78	.000	Significant
	Diabetic	40	124.00	11.138				
PPBS	Non Diabetic	40	116.05	7.282	17.885	78	.000	Significant
	Diabetic	40	232.40	40.494				
METS	Non Diabetic	40	7.85	3.534	.725	78	.471	Not Significant
	Diabetic	40	7.28	3.559				
ST Segment shift	Non Diabetic	40	1.6250	.57457	1.222	78	.225	Not Significant
	Diabetic	40	1.8125	.78191				
LVIDd	Non Diabetic	40	4.6175	.29862	.956	78	.342	Not Significant
	Diabetic	40	4.6900	.37540				
LVIDs	Non Diabetic	40	3.155	.5208	1.013	78	.314	Not Significant
	Diabetic	40	3.275	.5382				
LVEF	Non Diabetic	40	53.83	8.506	2.300	78	.024	Significant
	Diabetic	40	49.93	6.533				

The table displays the descriptive statistics of the sample size, mean, standard deviation and standard error. The table also shows that the t statistics, calculated as the ratio of the difference between sample means divided by the standard error of the difference. The column p value shows the probability value from the t distribution. Since the p value is greater than 0.05 for all the factors except FBS and PPBS from the above table, we accept the hypothesis. Hence there is no significant difference in the mean scores of the two groups of diabetic mellitus with respect to the systolic blood pressure, diastolic blood pressure, PR, Mets, ST Shifts, LVIDd, LVIDs. Since the p value is less than 0.05 it shows that there is significant difference in the mean scores of FBS and PPBS and LVEF among the diabetic and non diabetic patients.

ASSOCIATION BETWEEN CERTAIN FACTORS AND DIABETES MELLITUS

The association between the factors like Age, METS, ST Shifts, CAG, TMT and DM type were analyzed in this section. The chi square test is used at 5% level of significance.

Association between Diabetes mellitus and Age

The study patients were classified as diabetic and non diabetic and age of the patients were grouped in to four categories as up to 40 years, 41 – 50 yrs, 51 – 60 yrs and above 60 yrs.

The association between Age and Diabetes mellitus is analyzed and the results were given in the following bivariate Table 1.

Null Hypothesis: H_0**:** There is no association between **Age** and **Diabetes mellitus**

Table 1 Age * Diabetes mellitus

	DM Type		Total
	Non Diabetic	Diabetic	

Age	up to 40 yrs	3	3	6
		3.8%	3.8%	7.5%
	41 - 50	19	17	36
		23.8%	21.3%	45.0%
	51 - 60	15	14	29
		18.8%	17.5%	36.3%
	Above 60	3	6	9
		3.8%	7.5%	11.3%
Total		40	40	80
		50.0%	50.0%	100.0%

In order to find the relationship between the Age of the respondents and Diabetes mellitus, a chi-square test was used and result of the test is shown in the following table.2

Table .2 Age * Diabetes mellitus–Chi square Test

Age	Value	df	P value	Remark
Pearson Chi-Square	1.146	3	.766	Not Significant

It is noted from the above table that the ‘p; value is greater than 0.05 and hence the result is not significant at 5% level. Hence the hypothesis’ Age of the respondents and the Diabetes mellitus are not associated’ does hold well. From the analysis it is concluded that there is no close relationship between the Age of the respondents and Diabetes mellitus.

Association between Diabetes mellitus and METS

The association between METS and Diabetes mellitus is analyzed and the results were given in

the following bivariate Table 2.

Null Hypothesis: H_0**:** There is no association between **METS** and **Diabetes mellitus**

Table 3 METS * Diabetes mellitus

		DM Type		Total
		Non Diabetic	Diabetic	
METS	Severe	2	11	13
		2.5%	13.8%	16.3%
	Moderate	12	13	25
		15.0%	16.3%	31.3%
	Mild	26	16	42
		32.5%	20.0%	52.5%
Total		40	40	80
		50.0%	50.0%	100.0%

In order to find the relationship between the METS of the respondents and Diabetes mellitus, a chi-square test was used and result of the test is shown in the following table.4

Table 4 METS * Diabetes mellitus–Chi square Test

METS	**Value**	**df**	**P value**	**Remark**
Pearson Chi-Square	8.652	2	.013	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' METS of the respondents and

the Diabetes mellitus are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the METS of the respondents and Diabetes mellitus.

Association between Diabetes mellitus and ST Shifts

The association between ST Shifts and Diabetes mellitus is analyzed and the results were given in the following bivariate Table 5

Null Hypothesis: H_0**:** There is no association between **ST Shifts** and **Diabetes mellitus**

Table 5. ST Shifts * Diabetes mellitus

		DM Type		
		Non Diabetic	Diabetic	Total
ST Shift	Mild	18	10	28
		22.5%	12.5%	35.0%
	Moderate	20	21	41
		25.0%	26.3%	51.3%
	severe	2	9	11
		2.5%	11.3%	13.8%
Total		40	40	80
		50.0%	50.0%	100.0%

In order to find the relationship between the ST Shifts of the respondents and Diabetes mellitus, a chi-square test was used and result of the test is shown in the following table.6

Table 6 ST Shifts * Diabetes mellitus–Chi square Test

ST Shifts	Value	df	P value	Remark
Pearson Chi-Square	6.765	2	.034	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' ST Shifts of the respondents and the Diabetes mellitus are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the ST Shifts of the respondents and Diabetes mellitus.

Association between Diabetes mellitus and CAG

The association between CAG and Diabetes mellitus is analyzed and the results were given in the following bivariate Table 7.

Null Hypothesis: H_0**:** There is no association between **CAG** and **Diabetes mellitus**

Table 7 CAG * Diabetes mellitus

		DM Type		Total
		Non Diabetic	Diabetic	
CAG	N	5	2	7
		6.3%	2.5%	8.8%
	SVD	10	6	16
		12.5%	7.5%	20.0%
	DVD	18	12	30
		22.5%	15.0%	37.5%
	TVD	7	18	25
		8.8%	22.5%	31.3%
	Left main	0	2	2
		0.0%	2.5%	2.5%

Total	40	40	80
	50.0%	50.0%	100.0%

In order to find the relationship between the CAG of the respondents and Diabetes mellitus, a chi-square test was used and result of the test is shown in the following table.8

Table 8 CAG * Diabetes mellitus–Chi square Test

CAG	Value	df	P value	Remark
Pearson Chi-Square	10.326	4	.035	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' CAG of the respondents and the Diabetes mellitus are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the CAG of the respondents and Diabetes mellitus.

Association between Diabetes mellitus and TMT

The association between TMT and Diabetes mellitus is analyzed and the results were given in the following bivariate Table 9

Null Hypothesis: H_0**:** There is no association between **TMT** and **Diabetes mellitus**

Table 9 TMT * Diabetes mellitus

		DM Type		Total
		Non Diabetic	Diabetic	
TMT	Mild	26	16	42
		32.5%	20.0%	52.5%
	Moderate	12	13	25
		15.0%	16.3%	31.3%
	Severe	2	11	13
		2.5%	13.8%	16.3%
Total		40	40	80
		50.0%	50.0%	100.0%

In order to find the relationship between the TMT of the respondents and Diabetes mellitus, a chi-square test was used and result of the test is shown in the following table.10

Table 10 TMT * Diabetes mellitus–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	8.652	2	.013	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' TMT of the respondents and the Diabetes mellitus are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the TMT of the respondents and Diabetes mellitus.

Association between CAG and TMT

The association between TMT and CAG is analyzed and the results were given in the following bivariate Table 11.

Null Hypothesis: H_0**:** There is no association between **TMT** and **CAG**

Table 11 TMT * CAG

		CAG					Total
		N	SVD	DVD	TVD	Left main	
TMT	Mild	6	11	15	10	0	42
		7.5 %	13.8 %	18.8 %	12.5 %	0.0 %	52.5%
	Moderate	1	3	12	8	1	25
		1.3 %	3.8%	15.0 %	10.0 %	1.3 %	31.3%
	Severe	0	2	3	7	1	13
		0.0 %	2.5%	3.8%	8.8%	1.3 %	16.3%

Total	7	16	30	25	2	80
	8.8 %	20.0 %	37.5 %	31.3 %	2.5 %	100.0%

In order to find the relationship between the TMT of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.12

Table 12 TMT * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	28.285	8	.000	Highly Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' TMT of the respondents and the CAG are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the TMT of the respondents and CAG.

Association between DM TYPE and LVEF

The association between LVEF and DM TYPE is analyzed and the results were given in the following bivariate Table 13

Null Hypothesis: H_0**:** There is no association between **LVEF** and **DM TYPE**

Table 13 LVEF * DM TYPE

	DM Type		Total
	Non Diabetic	Diabetic	

LVEF_	Moderate	3	13	16
		3.8%	16.3%	20.0%
	Mild	21	18	39
		26.3%	22.5%	48.8%
	Normal	16	9	25
		20.0%	11.3%	31.3%
Total		40	40	80
		50.0%	50.0%	100.0%

In order to find the relationship between the LVEF of the respondents and DM TYPE, a chi-square test was used and result of the test is shown in the following table.14

Table 14 LVEF * DM TYPE–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	8.441	2	.015	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' LVEF of the respondents and the DM TYPE are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the LVEF of the respondents and DM TYPE.

Association between CAG and LVEF

The association between LVEF and CAG is analyzed and the results were given in the following bivariate Table 15

Null Hypothesis: H_0**:** There is no association between **LVEF** and **CAG**

Table 15 LVEF * CAG

Non Diabetic	CAG				Total
	N	SVD	DVD	TVD	

LVEF	Moderate	0	2	1	0	3
		0.0%	5.0%	2.5%	0.0%	7.5%
	Mild	1	3	12	5	21
		2.5%	7.5%	30.0 %	12.5 %	52.5%
	Normal	4	5	5	2	16
		10.0 %	12.5 %	12.5 %	5.0%	40.0%
Total		5	10	18	7	40
		12.5 %	25.0 %	45.0 %	17.5 %	100.0%

In order to find the relationship between the LVEF of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.16

Table 16 LVEF * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	9.361	6	.154	Not Significant

It is noted from the above table that the 'p; value is greater than 0.05 and hence the result is not significant at 5% level. Hence the hypothesis' LVEF of the respondents and the CAG are not associated' does hold well. From the analysis it is concluded that there is no close relationship between the LVEF of the respondents and CAG with respect to the non diabetic patients

The association between METS and CAG is analyzed and the results were given in the following bivariate Table 17

Null Hypothesis: H_0**:** There is no association between **METS** and **CAG**

Table 17 METS * CAG

Non Diabetic		CAG				Total
		N	SVD	DVD	TVD	
METS	Severe	0	2	0	0	2
		0.0%	5.0%	0.0%	0.0%	5.0%
	Moderate	1	3	6	2	12
		2.5%	7.5%	15.0 %	5.0%	30.0%
	Mild	4	5	12	5	26
		10.0 %	12.5 %	30.0 %	12.5 %	65.0%
Total		5	10	18	7	40
		12.5 %	25.0 %	45.0 %	17.5 %	100.0%

In order to find the relationship between the METS of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.18

Table 18 METS * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	9.486	6	.148	Not Significant

It is noted from the above table that the ‘p; value is greater than 0.05 and hence the result is not significant at 5% level. Hence the hypothesis’ METS of the respondents and the CAG are not associated’ does hold well. From the analysis it is concluded that there is no close relationship between the METS of the respondents and CAG with respect to the non diabetic patients

Association between CAG and ST SHIFTS

The association between ST SHIFTS and CAG is analyzed and the results were given in the following bivariate Table 19

Null Hypothesis: H_0**:** There is no association between **ST SHIFTS** and **CAG**

Table 19 ST SHIFTS * CAG

Non Diabetic		CAG				Total
		N	SVD	DVD	TVD	
ST Shift	Mild	4	4	7	3	18
		10.0 %	10.0 %	17.5 %	7.5%	45.0%
	Moderate	1	5	11	3	20
		2.5%	12.5 %	27.5 %	7.5%	50.0%
	severe	0	1	0	1	2
		0.0%	2.5%	0.0%	2.5%	5.0%
Total		5	10	18	7	40
		12.5 %	25.0 %	45.0 %	17.5 %	100.0%

In order to find the relationship between the ST SHIFTS of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.20

Table 20 ST SHIFTS * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	5.846	6	.441	Not Significant

It is noted from the above table that the 'p; value is greater than 0.05 and hence the result is not significant at 5% level. Hence the hypothesis' ST SHIFTS of the respondents and the CAG are not associated' does hold well. From the analysis it is concluded that there is no close relationship between the ST SHIFTS of the respondents and CAG with respect to the non diabetic patients

Association between CAG and LVEF

The association between LVEF and CAG is analyzed and the results were given in the following bivariate Table 21

Null Hypothesis: H_0**:** There is no association between **LVEF** and **CAG**

Table 21 LVEF * CAG

Diabetic	CAG					Total
	N	SVD	DVD	TVD	Left main	

LVEF	Moderate	0	1	3	9	0	13
		0.0%	2.5%	7.5%	22.5%	0.0%	32.5%
	Mild	0	2	6	9	1	18
		0.0%	5.0%	15.0%	22.5%	2.5%	45.0%
	Normal	2	3	3	0	1	9
		5.0%	7.5%	7.5%	0.0%	2.5%	22.5%
Total		2	6	12	18	2	40
		5.0%	15.0%	30.0%	45.0%	5.0%	100.0%

In order to find the relationship between the LVEF of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.22

Table 22 LVEF * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	16.715	8	.033	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' LVEF of the respondents and the CAG are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the LVEF of the respondents and CAG with respect to the Diabetic patients.

Association between CAG and METS

The association between METS and CAG is analyzed and the results were given in the

following bivariate Table 23

Null Hypothesis: H_0**:** There is no association between **METS** and **CAG**

Table 23 METS * CAG

Diabetic		CAG					Total
		N	SVD	DVD	TVD	Left main	
METS	Severe	0	0	3	7	1	11
		0.0%	0.0%	7.5%	17.5%	2.5%	27.5%
	Moderate	0	0	6	6	1	13
		0.0%	0.0%	15.0%	15.0%	2.5%	32.5%
	Mild	2	6	3	5	0	16
		5.0%	15.0%	7.5%	12.5%	0.0%	40.0%
Total		2	6	12	18	2	40
		5.0%	15.0%	30.0%	45.0%	5.0%	100.0%

` In order to find the relationship between the METS of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.24

Table 24 METS * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	16.715	8	.033	Significant

It is noted from the above table that the ‘p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis’ METS of the respondents and

the CAG are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the METS of the respondents and CAG with respect to the Diabetic patients

Association between CAG and ST SHIFTS

The association between ST SHIFTS and CAG is analyzed and the results were given in the following bivariate Table 25

Null Hypothesis: H_0**:** There is no association between **ST SHIFTS** and **CAG**

Table 25 ST SHIFTS * CAG

Diabetic		CAG					Total
		N	SVD	DVD	TVD	Left main	
ST Shift	Mild	2	4	1	3	0	10
		5.0 %	10.0 %	2.5%	7.5%	0.0%	25.0%
	Moderate	0	2	7	12	0	21
		0.0 %	5.0%	17.5 %	30.0 %	0.0%	52.5%
	severe	0	0	4	3	2	9
		0.0 %	0.0%	10.0 %	7.5%	5.0%	22.5%

Total	2	6	12	18	2	40
	5.0 %	15.0 %	30.0 %	45.0 %	5.0%	100.0%

In order to find the relationship between the ST SHIFTS of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.26

Table 26 ST SHIFTS * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	22.323	8	.004	Significant

It is noted from the above table that the 'p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis' ST SHIFTS of the respondents and the CAG are not associated' does not hold well. From the analysis it is concluded that there is close relationship between the ST SHIFTS of the respondents and CAG with respect to the Diabetic patients

Association between CAG and TMT

The association between TMT and CAG is analyzed and the results were given in the following bivariate Table 27

Null Hypothesis: H_0**:** There is no association between **TMT** and **CAG**

Table 27 TMT * CAG

Non Diabetic		CAG				Total
		N	SVD	DVD	TVD	
TMT	Mild	4	5	12	5	26
		10.0 %	12.5 %	30.0 %	12.5 %	65.0%
	Moderate	1	3	6	2	12
		2.5%	7.5%	15.0 %	5.0%	30.0%
	Severe	0	2	0	0	2
		0.0%	5.0%	0.0%	0.0%	5.0%
Total		5	10	18	7	40
		12.5 %	25.0 %	45.0 %	17.5 %	100.0%

In order to find the relationship between the TMT of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.28

Table 28 TMT * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	6.810	6	.339	Not Significant

It is noted from the above table that the ‘p; value is greater than 0.05 and hence the result is not significant at 5% level. Hence the hypothesis’ TMT of the respondents and the CAG are not associated’ does hold well. From the analysis it is concluded that there is no close relationship between the TMT of the respondents and CAG with respect to the Diabetic patients.

Association between CAG and TMT

The association between TMT and CAG is analyzed and the results were given in the following bivariate Table 29

Null Hypothesis: H_0**:** There is no association between **TMT** and **CAG**

Table 29 TMT * CAG

Diabetic		CAG					Total
		N	SVD	DVD	TVD	Left main	
TMT	Mild	2 5.0 %	6 15.0 %	3 7.5%	5 12.5 %	0 0.0 %	16 40.0%
	Moderate	0 0.0 %	0 0.0%	6 15.0 %	6 15.0 %	1 2.5 %	13 32.5%
	Severe	0 0.0 %	0 0.0%	3 7.5%	7 17.5 %	1 2.5 %	11 27.5%
Total		2 5.0 %	6 15.0 %	12 30.0 %	18 45.0 %	2 5.0 %	40 100.0%

In order to find the relationship between the TMT of the respondents and CAG, a chi-square test was used and result of the test is shown in the following table.30

Table 30 TMT * CAG–Chi square Test

	Value	df	P value	Remark
Pearson Chi-Square	16.715	8	.033	Significant

It is noted from the above table that the ‘p; value is less than 0.05 and hence the result is significant at 5% level. Hence the hypothesis’ TMT of the respondents and the CAG are not associated’ does not hold well. From the analysis it is concluded that there is close relationship between the TMT of the respondents and CAG with respect to the Diabetic patients

DISCUSSION

Coronary artery disease in patients with diabetes is a rising scourge in developing and underdeveloped countries. It remains the most common single cause of mortality and morbidity in men below 65 years of age. For early diagnosis of CAD, before the occurrence of major mishap like myocardial infarction, treadmill stress test remains a chief, cost effective and widely available and applicable approach. The advent of selective coronary arteriography has enabled the clinician to correlate in vivo coronary artery anatomy with such non-invasive tests as exercise electrocardiography.

Numerous recent reports using coronary angiography have placed into better performance. The role of such stress testing in the evaluation and detection of patients with potential coronary artery disease. However, many of the studies have interpreted the exercise electrocardiogram in diabetics solely as normal or abnormal in the arbitary basis of a predetermined extent of ischaemic ST segment depression. None of the methods of exercise stress testing permits a separation between normal subject and patients with coronary artery disease. Therefore we believe that the most valid diagnose is the application of exercise ECG would be to determine the probablity of the maximal extent of the ST segment depression, being association with coronary artery disease in patients with diabetes.

In this study, we included all the patients with the positive exercise treadmill test to determine how will the existence of three vessel or LMS can be predicted among patients referred for a treadmill exercise test with suspected CAD.

Correlation between CAG & TMT

When we analysed the relationship of CAG with TMT, we found that there was statistically significant correlation between CAD and TMT. In diabetics and non-diabetics (P = 0.000 highly

significant) from our study, TMT correlation with normal epicardial coronary arteries was 8.8%, whereas for single vessel disease, double vessel disease & TVD and left main disease were 20%, 37.5%, 31.3 and 2.5% respectively.

Our findings were similar to that of **Roitman etal finding**[15], in their study, they found that strong correlation between positive exercise treadmill test and CAG. **Proudfit etal**[30] reported that METS in an independent predictor of the exercise of TVD or LMS when we analysed the relationship between METS and diabetes mellitus, we found that METS < 5 was 13.8%. in diabetic and only 2.5% in non-diabetic, and we also noted that METS > 7 was 20% in diabetic and 32.5% in non diabetic. Our findings were similar to that of **Bortel AG etal** [13]who reported a positive relationship between workload and existence of TVD or LMS in diabetics and non-diabetics. We also noted that METS < 5 was 17.5% in TVD and 7.5% in DVD and 2.5% in LMS in diabetic patient but only 10/% in TVD, 5% in DVD and 7.5% in SVD in non-diabetics.

Saeed Sadegan MD etal[10] reported that workload equal to or less than 7 METS indicated on 8.3% chance of detecting LMS confirmed by angiographic findings. In our study, there is 5% chance of detecting LMS which was confirmed by CAG. In contrast, when the workload was raised to greater than 7 METS, this chance decreased to 0%.

When analysing the relationship between ST shifts and CAG, we found that there is close relationship between ST segment shift of the respondents and CAG with respect to the diabetic group (P = 0.004). From our study, we noted that ST segment > 2 mm in CAD patients with diabetes, was 10% in DVD, 7.5% in TVD and 5% in LMS. Whereas in non-diabetics 5% in SVD, 5% in DVD, 7.5% in TVD and 0% in LMS. Our findings were similar to that of **Alan G.Bortel etal**[13] who reported that the degree of ST Segment depression was related to the severity of CAG. They found that patients having ST segment depression greater than 2.00 mm were much more likely to have TVD, than those with less severe

ST depression. These findings correlate with our study.

Although the occurrence of further ST-T depression with exercise was strongly correlated with the presence of CAD in patients with diabetes, in our study, the degree of ST-T depression was not correlated with the extent of coronary artery obstruction, seen on angiography. This lack of quantitative correlation was also reported by **Cohn and Roitman etal**[15]. A possible recent for lack of correlation in our study is the fact that additional ST segement depression was one of the indication for stopping the test, thus restricting the amount of ST depression which developed during exercise.

Roitman etal[15] also suggested that patients who developed more than or 1 mm additional ST-T depression with exercise had a significantly lower ratio of peak heart rate to target heart rate than those who did not (0.82 and 0.95 respectively, $P < 0.001$). Since peak heart rate is shown to be lower in CAD patients, it follows that additional ST depression is closely correlated with the presence of CAD.

Two of our patients who showed further ST-T depression, yet one had normal CAG and another one had SVG were females. Our retrospective enquiry into one of these patients was receiving digoxin until five days prior to the exercise test, which could account for a false positive additional ST-T depression of 2 mm on exercise. Our one "false positive" responder in women and others also have reported this phenomenon. **Kawn B etal**[38] coming have reported a lower diagnostic value of pre exercise ST-T changes in women than in men, when all patients resting ECGs were normal[33].

We found in our study that there was statistically significant correlation between LV dysfunction and diabetes Mellitus type. We observed that there was significant LV Dysfunction 16.3% in diabetic patients and 3.5% in non-diabetic patients ($P = 0.015$). Our study demonstrated that significant LV dysfunction in diabetic patient group was present in 7.5% in SVD, 7.5% in DVD, 22.5% in TVD, ($P =$

0.033) whereas in non-diabetic patients group it was only 5% in SVD, 2.5% in DVD and none in TVD, (P = 0.154).

Our findings were similar to that of **Jaishankar etal finding**[5], in that study they found that 27.5% of the patients with SVD had LVD (P < 0.001) in the diabetic group, whereas only21.2% of the non-diabetic group had significant LVD. **Bagchi Soumita etal**[9] also demonstrated that significant LVD was statistically significant in patients with diabetes (P < 0.05). From our analysis, it is concluded that there is close relationship between the LVEF of the respondents and CAG with respect to the diabetic patients (P=0.015).

Among diabetic patients, 6 (7.5%) had chronic total occlusion involving SVD and 12 (15%) had DVD 18 (22.55%) had TVD chronic total occlusion. The corresponding values in non-diabetics was 10 (12.5%) 18 (22.5%) and 7 (8.8%) respectively (P = 0.035). When we analysing diffuse lesion in diabetic patients it was 32 (80%) and 18 (45%) in non-diabetics. Our findings correlated with that of **Srikala etal findings**[54], in their study, they were observed diffuse lesion were present in 47% in diabetics and 24% in non-diabetics. **Bagchi Saumati etal**[9] also demonstrated that significantly higher incidence of severe disease (DVD, TVD) was found in CAD patients with diabetes (P < 0.05).

From our study, we have observed that the pattern of vessel involvement was similar in both groups with mostly commonly involved was left anterior descending artery. Our findings correlate with **Bagchi Saumati etal observation**[9].

When we are analysing LMCA involvement in patients with CAD with diabetes and non-diabetes, we found that LMCA involvement was higher in diabetes patients (2.5%) versus none in non-diabetics patients (P = 0.000). This finding correlated with that of **Srikala etal study**, in their study they

demonstrated that LMCA involvement was higher in diabetic patients ($P < 0.001$).

When we are analysing bifurcation lesion in both groups, we found that Bifurcation lesion was higher in diabetics (12%) and in non-diabetics, it was only 2%. We also noted that calcified lesions were 32% in diabetics and 14% in non-diabetics. Our findings correlates with that of **Srikala etal study**[54], in their study, they observed that bifurcation lesions were 43% in diabetics and 12% in non-diabetics and calcified lesions were 68% in diabetics and 38% in non-diabetics.

LIMITATIONS

Potential limitation of this study include the relatively small sample size and low absolute number of study end points. In future investigation, more patients should be studied by the previous method.

The preferential selection of patients based on an abnormal exercise test response for referral to CAG to verify CAD ("work up bias") may be seen as an apparent increase in sensitivity and decreased specificity in this study.

Finally, the exclusion of women from this study confines its conclusion to men.

CONCLUSION

1. In the present study, there is strong correlation between positive exercise treadmill test and coronary angiographic profile in patients with diabetes and non-diabetes.

2. METS is an independent predictor of the existence of the Three Vessel or Left Main Disease.

3. It is shown that in our study that there is positive treadmill test in METS less than 5 predicts an increased likelihood of TVD (27.5%) and LMS (2.5%) in patient with diabetes.

4. Our study confirmed that there is a positive relationship between workload and existence of TVD or LMS.

5. Our data clearly support the concept that workload at which the ischaemic changes are determined is important in the evaluation of the severity of the disease.

6. This study shows that the degree of ST segment depression correlates well with the angiographic degree of the stenosis.

7. ST segment depression > 2.00 mm has significant correlation to predict TVD (7.5%) and LMS (5%).

8. It is shown that in our study that there is statistically significant correlation between the extent of LV dysfunction and TVD (22.5%) in patients with diabetes.

9. Diabetes Mellitus results in a higher incidence of chronic total occlusion and more proximal and more diffuse coronary lesion (80%).

10. Chronic total occlusion involving TVD in diabetic (22.5%) is more than in patients without diabetes (8.8%).

11. Complex lesion like Bifurcation (12%) and calcification (32%) was significantly higher in diabetics.

12. LMCA disease involvement was higher in diabetics, when compared with non-diabetics i.e. 2.5% in diabetics.

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PROFORMA

A COMPARATIVE STUDY OF CORONARY ANGIOGRAPHIC PROFILE WITH POSITIVE EXERCISE TREADMILL TEST IN PATIENTS WITH DIABETES AND NON-DIABETES

Name : Age : Sex :

Occupation : CD No : Address :

Risk Factor Profile : Diabetes Hypertension Smoking
Obesity Hyperlipidemia Previous CAD

CLINICAL PROFILE :

Chest Pain CCS Class

Vital Parameters PR :

BP :

CVS :

RS :

Other Systems

Drugs used

INVESTIGATIONS :

Blood Sugar : Fasting Postprandial

Cardiac Enzymes : CPK - CPKMB –

Lipid Profile :

ECG :

CHEST x-ray PA view :

TREADMILL TEST :

Exercise Time : Protocol : METS :

MAX HR : % THR : RFT -

THR achieved Normal HR / BP Response.

Significant ST-T changes

ANGINA / ARRYTHMIA

RECOVERY PERIOD :

IMPRESSION :

POSITIVE FOR INDUCIBLE ISCHAEMIA - MILD
MODERATE
STRONGLY POSITIVE

ECHOCARDIOGRAPHY

Systolic function : LV Dimension : LVIDd :

LVIDs : EDV : ESV : EF : FS :

RWMA : Wall Thickness :

Diastolic function : TRANSMITRAL FLOW PATTERN E A

CORONARY ANGIOGRAM

Technique :

Artery used :

Catheter used :

Dye used :

Aortic Systolic :

Diastolic :

Mean :

LV Systolic :

Diastolic :

LMCA :

LAD :

LCX :

RCA :

LIMA :

RENAL ARTERIES :

DIAGNOSIS :

GLOSSARY

CAD	:	Coronary Artery disease
DM	:	Diabetes Mellitus
ECG	:	Electro cardio Gram
LMS	:	Left Main Stenosis
MI	:	Myocardial Infarction
CVD	:	Cardio Vascular Disease
LDL	:	Low Density Lipoprotein
VLDL	:	Very Low Density Lipoprotein
AGE	:	Advanced Glycation End Product
LVEF	:	Left Ventricular Ejection Fraction
LVEDV	:	Left Ventricular End Diastolic Volume
LVESV	:	Left Ventricular End Systolic Volume
LVEDD	:	Left Ventricular End Diastolic Dimension
LVESD	:	Left Ventricular End Systolic Dimension
TMT	:	Tread Mill Test
LMCA	:	Left Main Coronary Artery
LAD	:	Left Anterior Descending Artery
LCX	:	Left Circumflex Artery
RCA	:	Right Coronary Artery
METS	:	Metabolic Equivalent
CCS	:	Canadian Cardio Vascular Society
SVD	:	Single Vessel Disease
DVD	:	Double Vessel Disease
TVD	:	Three Vessel Disease